REFERENCES

1. Habets JMW, Tank B, Vuzeuski VD, van Reede EC, Stolz E, van Joost T: Characterization of the mononuclear infiltrate in basal cell carcinoma; a predominantly T cell-mediated immune response with minor participation of Leu 7 + (natural killer) and Leu 14 + (B) cells. J Invest Dermatol 90:239–292, 1988
2. Manara GC, Ferrari C, DePanfilis G: HNK-1 antigen is not specific for natural killer cells. J Invest Dermatol 91:374, 1988
3. Tank B, Habets JMW, van Joost T: HNK-1 antigen is not specific for natural killer cells—reply. J Invest Dermatol 91:374–375, 1988
4. Hercend T, Schmidt TE: Characteristics and uses of natural killer cells. Immunol Today 9:291–293, 1988
5. Moy PM, Holmes EC, Golub SH: Depression of natural killer cytotoxic activity in lymphocytes infiltrating human pulmonary tumours. Cancer Research 45:57, 1985

Dual Genotype in Cutaneous T Cell Lymphoma: Immunoglobulin Gene Rearrangement in Clonal T Cell Malignancy

To the Editor:

In their study, Berger et al [1] reported a surprisingly high incidence of dual genotype in cutaneous T cell lymphomas (CTCL). Four of 13 cases in leukemic phase presented both a TCR and an IgJH rearrangement in the blood.

We should like to raise a point about this interesting article by a comparison with our own results. Like Whittaker [2], we failed to find an additional JH rearrangement to TCR gene rearrangement in peripheral blood of Sézary syndrome, and we were surprised by the absence of any genotypic study in skin lesions.

We studied 20 skin biopsies of CTCL with both JH and beta TCR gene probes. We also found a high incidence of bigenotype: 5 cases (20%) had both a TCR and IgJH rearrangement. Thus, the skin samples confirmed the results obtained from the blood studies. But, unlike Berger, in one case we had more than a single JH allele rearranged.

Berger et al presented three hypotheses for dual genotype in CTCL but our results suggest that there is a fourth explanation: The pathology may not be monoclonal but oligoclonal. It is well-known that, in some cases, there are more than two rearranged bands suggesting B cell biclonality [3] or different T cell clones in separate skin lesions from the same patient [4]. One of our cases agreed with the oligoclonality theory. Two skin biopsies from the same patient were studied; the second biopsy had, besides the same rearranged IgJH band, two additional rearranged bands, although they had the same genotype with beta and gamma TCR gene probes.

We should be interested to know if the authors have eliminated this explanation for the cases in which they did not perform genotypic study on T cell enrichment of peripheral blood lymphocytes.

Y.J. Bignon, M.D.
P. Souteyrand, M.D.
H. Roger, M.D.
D. Bernard, Ph.D.
Centre Jean Perrin (Y.J.B., D.B.) and Service de Dermatologie Hôtel Dieu (P.S., H.R.), Clermont-Ferrand, France

REFERENCES

1. Berger CL, Eisenberg A, Soper L, Chow J, Simone J, Gapas Y, Cacciapaglia B, Bennett L, Edelson RL, Warburton D, Benn P: Dual genotype in cutaneous T cell lymphoma: immunoglobulin gene rearrangement in clonal T cell malignancy. J Invest Dermatol 90:73–77, 1988
2. Whittaker SJ: Dual genotype in cutaneous T cell lymphoma: immunoglobulin gene arrangement in clonal T cell malignancy. J Invest Dermatol 91:192, 1988
3. Sklar J, Cleary ML, Thielemans K, Gralow J, Warnke RA, Levy R: Biclonal B cell lymphoma. N Engl J Med 311:20–27, 1984
4. Weiss LM, Wood GS, Trela M, Warnke RA, Sklar J: Clonal T cell populations in lymphomatoid papulosis: evidence of a lymphoproliferative origin for a clinically benign disease. N Engl J Med 315:475–479, 1986

REPLY

We have read the letter by Drs. Bignon et al concerning our manuscript entitled "Dual genotype in cutaneous T cell lymphoma: Immunoglobulin gene rearrangement in clonal T cell malignancy" by Berger et al (J Invest Dermatol 90:73–77, 1988). We would like to respond to the points raised in this letter.

We did not study the skin lesions of these patients as the goal of our investigation was to confirm leukemic disease dissemination in already diagnosed patients by DNA hybridization and compare this technology with previously developed methods. We were interested to learn that skin samples studied by Dr. Bignon and associates also contained a dual genotype. We are surprised that this phenomenon was not detected in the peripheral blood of these patients but we do not know the stage of their disease at the time of the study. It may be that these cells were sequestered in the skin at an early stage of disease progression and may enter the periphery at a later time point.

Dr. Bignon and colleagues also suggest an oligoclonal origin for some cases of cutaneous T cell lymphoma (CTCL) based on the presence of additional JH rearrangements found in the skin biopsy of one patient. The presence of additional rearrangements detected with the JH probe could reflect clonal evolution involving the second allele, deletion of heavy-chain gene sequences, or other complex exchanges. It is equally possible that this JH rearrangement represents a dual neoplasm as the authors apparently did not confirm that these rearrangements were restricted to the clonal T cell population. Although some cases of CTCL may be oligoclonal in origin we have demonstrated no evidence to support this contention in our studies and have confirmed monoclonality by karyotypic analysis in several cases.

We believe further studies are required to fully elucidate the nature of the transformational event in CTCL and the origin of this disease.

Carole L. Berger, Ph.D.
Peter Benn, Ph.D.
Arthur Eisenberg, Ph.D.
Laurie Soper, Ph.D.
Lisa Bennett, B.S.
Barbara Cacciapaglia, B.S.
Department of Dermatology, Columbia University, New York City, New York
Lifecodes Corporation, Valhalla, New York